Correspondence

The Editorial Board will be pleased to receive and consider for publication correspondence containing information of interest to physicians or commenting on issues of the day. Letters ordinarily should not exceed 600 words, and must be typewritten, double-spaced and submitted in duplicate (the original typescript and one copy). Authors will be given an opportunity to review any substantial editing or abridgement before publication.

Hyperbaric Oxygen for Carbon Monoxide Poisoning

TO THE EDITOR: The fine article in the November 1981 issue by Dennis Landers, MD, PHD, on carbon monoxide poisoning[1] deserves some comment. In the article he shows that emission control systems on automobile exhausts have decreased the possibility of death by carbon monoxide poisoning during suicide attempts—an unexpected environmental bonus, indeed.

Although the thrust of the article is not toward treatment, he does mention oxygen therapy and neglects the most impressive modality—hyperbaric oxygen. He implies that the only treatment available is high flow oxygen to hasten the dissociation of carboxyhemoglobin. Hyperbaric oxygen will do this more rapidly, will increase the flow of oxygen to the hypoxic tissues and will decrease the cerebral edema that can cause central nervous system sequelae.[2]

This is being written to remind physicians that there are many facilities available for hyperbaric oxygen therapy. Many are related to scuba diving, some are associated with hospital therapeutic and research units. The location of the closest hyperbaric chamber can be obtained by calling the Diving Accident Network which is associated with Duke University Medical Center. The number is manned 24 hours a day. It is (919) 684-8111. In an emergency, collect calls will be accepted. Forgetting hyperbaric oxygen therapy can lead to needless morbidity and mortality.

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REFERENCES

1. Landers D: Unsuccessful suicide by carbon monoxide: A secondary benefit of emissions control. West J Med 1981 Nov; 135:360-363
2. Anderson GK: Treatment of carbon monoxide poisoning with hyperbaric oxygen. Milit Med 1978 Aug; 143:8

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TO THE EDITOR: We enjoyed reading the interesting case of carbon monoxide poisoning recently reported by Landers[1]; however, his comment that carbon monoxide has "little in the way of a true toxic effect" is not quite accurate. Actually, carbon monoxide (CO) poisoning is a complex phenomenon that involves both direct and indirect toxic effects.

As noted by Landers, CO causes tissue hypoxia by disturbing blood oxygen transport. This indirect toxic effect results from the preferential affinity of CO for hemoglobin and the associated leftward shift of the oxy-hemoglobin dissociation curve. These effects are thoroughly discussed in standard references.

Less well recognized is the fact that CO also is directly toxic to the cellular cytochrome system,[2-6] with inhibition of cytochrome A_3 oxidase being the most important effect. Although there is little information available about the exact mechanism and pharmacokinetics of CO-cytochrome binding, it is believed that these direct toxic effects on cellular respiration account for the often noted disparity between carboxyhemoglobin (COHb) levels and a patient's clinical condition.

Illustrative of the direct toxicity of carbon monoxide are the six patients recently reported by Myers and co-workers,[7] in whom the COHb levels were minimal or zero but who were still profoundly symptomatic from CO poisoning. All of these patients failed to respond to standard therapy but promptly recovered following treatment with hyperbaric oxygen (HBO). We have had some similar experience.

The usefulness of and rationale for treating CO poisoning with HBO have been discussed elsewhere.[5,8-11] Among the beneficial effects are a greatly diminished carboxyhemoglobin half-life, enhanced tissue clearance of residual CO, reduced cerebral edema and reversal of the cytochrome oxidase inhibition. The specific mechanism by which HBO overcomes the cytochrome toxicity in both CO and cyanide poisoning has not been well delineated.

At present it is recommended that anyone who is symptomatic and has a COHb level of 25 percent or greater should be treated with HBO.[5] However, the history of exposure and the overall clinical appearance of the patient should be the prime determinants of who is referred for HBO regardless of the actual COHb level.

It is important that emergency physicians and other personnel involved in the care of these types of cases establish liaison with the HBO treatment facilities in their communities so that referral to such facilities can be expeditiously accomplished when needed. Contrary to what Coleman noted in her recent excellent review of smoke inhalation[12] —that is, that HBO is "seldom available in time to be beneficial" in cases of CO poisoning—HBO treatment is now readily available in many metropolitan areas. (For example, there are five hospitals in the San Francisco Bay Area which currently have one or more monoplace recompression chambers and additional facilities are expected to become available soon.) Also, as noted by Myers,[7] severely poisoned patients may have dramatic recoveries even when HBO treatment is delayed many hours. The importance of knowing the availability of HBO treatment has been further underscored by recent litigation in which large settlements were awarded to plaintiffs because they were not referred for HBO treatment when facilities for such were locally available.

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REFERENCES

1. Landers D: Unsuccessful suicide by carbon monoxide: A secondary benefit of emission control. West J Med 1981 Nov; 135:360-363
2. Goldbaum LR, Orellano T, Degal E: Mechanism of the toxic action of carbon monoxide. Ann Clin Lab Sci 1976; 6:372-376
3. Chance B, Erecinska M, Wagner M: Mitochondrial responses to carbon monoxide toxicity. Ann NY Acad Sci 1970; 174:193-204
4. Goldbaum LR, Ramirez RG, Absalon KB: What is the mechanism of carbon monoxide toxicity? Aviat Space Environ Med 1975; 46:1289-1291
5. Kindwall EP: Carbon monoxide poisoning and cyanide poisoning. HBO Rev 1980; 1:115-122
6. Somogyi E, Balogh I, Rubanyi G, et al: New findings concerning the pathogenesis of acute carbon monoxide (CO) poisoning. Am J Forensic Med Path 1981; 2:31-39
7. Myers RAM, Snyder SK, Linberg S, et al: Value of hyperbaric oxygen in suspected carbon monoxide poisoning. JAMA 1981; 246:2478-2480
8. Smith G, Sharp GR: Treatment of carbon monoxide poisoning with oxygen under pressure. Lancet 1960 Oct 22; 2:905-906
9. Sluijter ME: The Treatment of Carbon Monoxide Poisoning by Administration of Oxygen at High Pressure. Springfield, IL, Charles C Thomas, 1963
10. Kindwall EP: Carbon monoxide and cyanide poisoning, *In* Davis JC, Hunt TK (Eds): Hyperbaric Oxygen Therapy. Bethesda, MD, Undersea Medical Society, Inc, 1977, pp 177-190
11. Myers RAM, Linberg SE, Crowley RA: Carbon monoxide poisoning: The injury and its treatment. JACEP 1979; 8:479-484
12. Coleman DL: Smoke inhalation—Medical Staff Conference, University of California, San Francisco. West J Med 1981 Oct; 135-300-309

Impaired Cognitive Judgment in Relation to the Chronic Use of Anticoagulants

TO THE EDITOR: Last year, a corporation president asked about a possible relationship between anticoagulants and impaired judgment. He had noted that, in several instances, otherwise very competent and experienced senior executives had made surprisingly poor decisions or had exercised remarkably bad judgment. (Even the executives themselves, all men, were appalled and confused by their actions.) Subsequently, it had been discovered that, in every case, the individual executive was taking prescribed anticoagulant medication at the time of judgmental errors.

Cursory inquiry of area physicians suggested no such relationship or knowledge of the correlation. Several other major corporation leaders were contacted and asked to view this issue retrospectively. They were asked to determine what significant examples of poor judgment, if any, had occurred in their companies in the recent years. Nine such incidents were identified and, in reviewing the medical histories of the individuals responsible, six were shown to have been taking anticoagulant medications at the time of the inappropriate actions.

Concurrently, I discovered three of my clients who were experiencing frequent bouts of impotency were using only one medication: sodium warfarin (Coumadin, Panwarfin). In addition, two Air Force officers and five businessmen who were either then taking this same drug or had used it in the past made statements such as, "I felt strangely vulnerable and mentally uncertain," and, "It was like not having both oars in the water." Another said, "Being 63, I wondered if I was already getting senile. I couldn't remember whether I'd actually talked to someone or just thought about it." All expressed concern over resulting sexual dysfunctions. Two indicated more of a reduced libidinal drive as the cause of their unexpected lack of sexual activity rather than the inability to properly function. They reported masturbation not to be a problem while the others claimed they had not attempted it since the onset of the difficulties. (This therapist has never encountered an impotency case before or since these cases in which masturbation had not been explored by the client.) None had ever been tested by a nocturnal tumescence device (NTD) nor had any consulted a urologist, neurologist or therapist.

Another patient, a 64-year-old medical doctor, was already falling into disfavor with colleagues due to occasional surgical errors or poor work before a stroke forced his retirement. These errors reportedly occurred during operations when he needed to react quickly and accurately. In several cases, other surgeons actually had to do postoperative repair work. This had been unheard of